Bilateral Petrous Ridge Dural Arteriovenous Malformations Treated by a Combination of Endovascular Embolization and Surgical Excision

A Case Report

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Summary

We report the first case of bilateral complex petrosal arteriovenous malformations (AVM). The patient was treated by a combination of transarterial embolization and surgical excision on the right-sided AVM, and by transarterial and transvenous embolization on left-sided lesion.

Introduction

Arteriovenous malformations (AVMs) of the dura mater usually occur in or near the dural walls of venous sinuses. The cavernous sinus and transverse-sigmoid sinus are most frequently involved. A dural AVM of the superior ridge of the petrous bone is rare. Its shunt is located at the deflection of the dura of the tentorium at the top of the petrous bone where the petrosal vein connects with the superior petrosal sinus. Unlike dural AVMs of the transverse-sigmoid or cavernous sinuses that drain mostly into the venous sinuses, all petrous dural AVMs drain initially directly into pial veins, causing aggressive clinical presentations [1-10]. Increased venous outflow in the pial veins induces venous hypertension, then eventually neurological dysfunctions. Like other dural AVMs with pial venous drainage, these may produce venous aneurysms and subarachnoid hemorrhage [3,9-10]. Therefore, it is essential to diagnose these dural AVMs early and treat them properly. But, unfortunately, deep-seated dural AVMs are among the most difficult ones to cure by endovascular means alone, since arterial feeders are multiple and the basal tentorial artery of the internal carotid artery often contributes to the supply. Surgical intervention becomes necessary to eliminate AV shunting if endovascular embolization does not close completely.

We report a very rare case of bilateral complex petrosal dural AVMs treated by a combined therapy of endovascular embolization and surgery.

Case Report

History and Examination

A 55-year-old man presented with progressive right-sided numbness for six months. The patient had first developed a decreased sensation to light touch in his right lower leg. This slowly progressed to the thigh and then to his torso and right shoulder. Sensory change expanded to his face and head, eventually to the right arm. At this point, his hand was relatively spared but, otherwise, he had decreased sensation throughout the entire right side of his body. There was no weakness. There was no loss of bowel or bladder control, no difficulty in swallowing, no changes in voice or vision. He

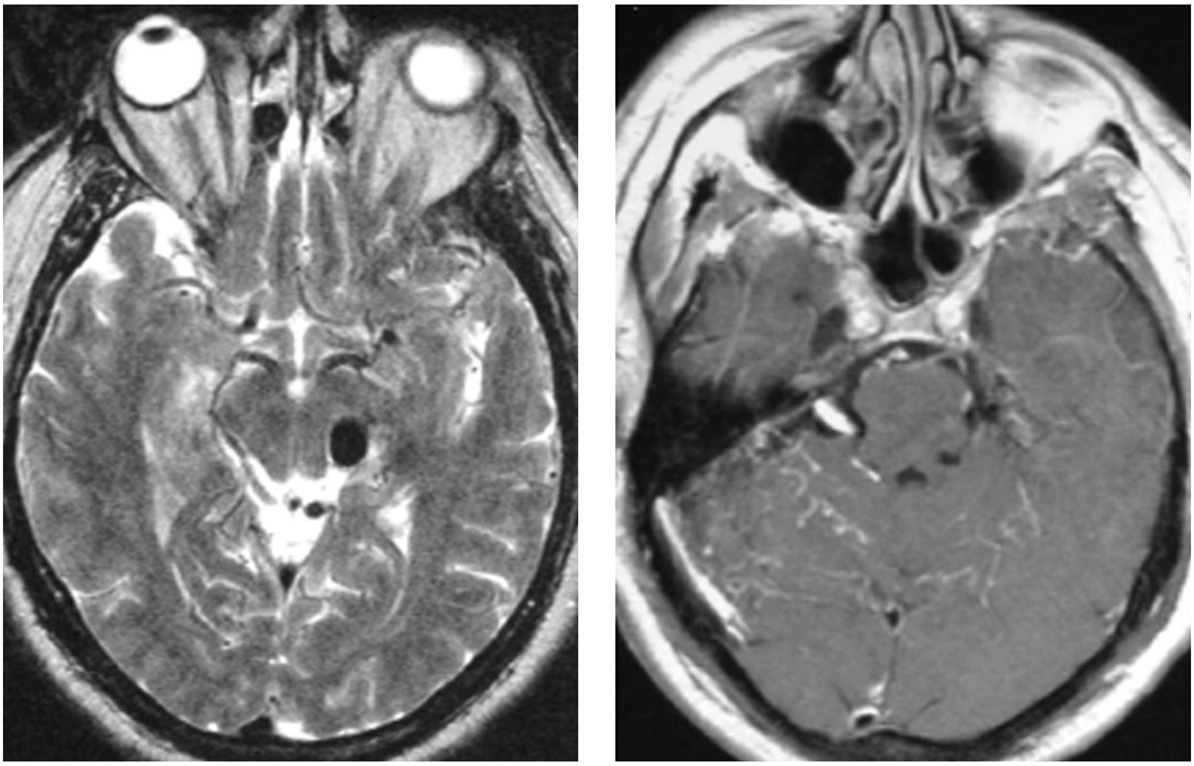

Figure 1 A) T2-weighted axial magnetic resonance (MR) image shows a venous aneurysm in the left perimesencephalic cistern which is indenting the left midbrain. B) Gadolium-enhanced T1-weighted axial image shows a venous aneurysm in the right cerebellopontine angle near the trigeminal ganglion and multiple dilated serpiginous dilated vessels in the cerebellar hemispheres bilaterally which are clustered in the region of the tentorium.

did not have any significant medial history or trauma.

At the time of initial evaluation, he did not have any significant physical or neurological signs except decreased sensation to light touch along the entire right side of the body, sparing the right hand.

Radiographic Studies

Magnetic resonance (MR) images showed a giant vascular sac in the left perimesencephalic cistern, indenting the left midbrain and extending superiorly into the left thalamus (figure 1A). There was another vascular sac in the right cerebellopontine angle near the trigeminal ganglion and multiple serpiginous dilated vessels were seen on the surface of the cerebellar hemispheres bilaterally, clustered in the region of the tentorium (figure 1B).

The left side angiogram was done first. It demonstrated AV shunting located at the left petrous ridge fed by the petrosal and petrosquamous branches of the middle meningeal artery, transmastoid branches of the occipital artery and hypoglossal branch of the ascending pharyngeal artery as well as the basal tentorial branches of the internal carotid artery. The initial venous drainage was through the petrosal vein, which then drained into the lateral mesencephalic and posterior mesencephalic veins, eventually to the vein of Galen and to the straight sinus. The proximal portion of the basal vein of Rosenthal was filled by retrograde flow. There was a giant aneurysm at the junction of the lateral mesencephalic and the posterior mesencephalic veins.

The right side angiogram was followed. A similar dural shunting was found at the mirror image location, petrous ridge. The arterial supplies were from the basal tentorial branch of the internal carotid artery, petrosal and petrosquamosal branches of the middle meningeal artery, artery of foramen rotundum, the hypoglossal branch of the ascending pharyngeal artery, transmastoid branch of the occipital artery and tentorial branches of the right

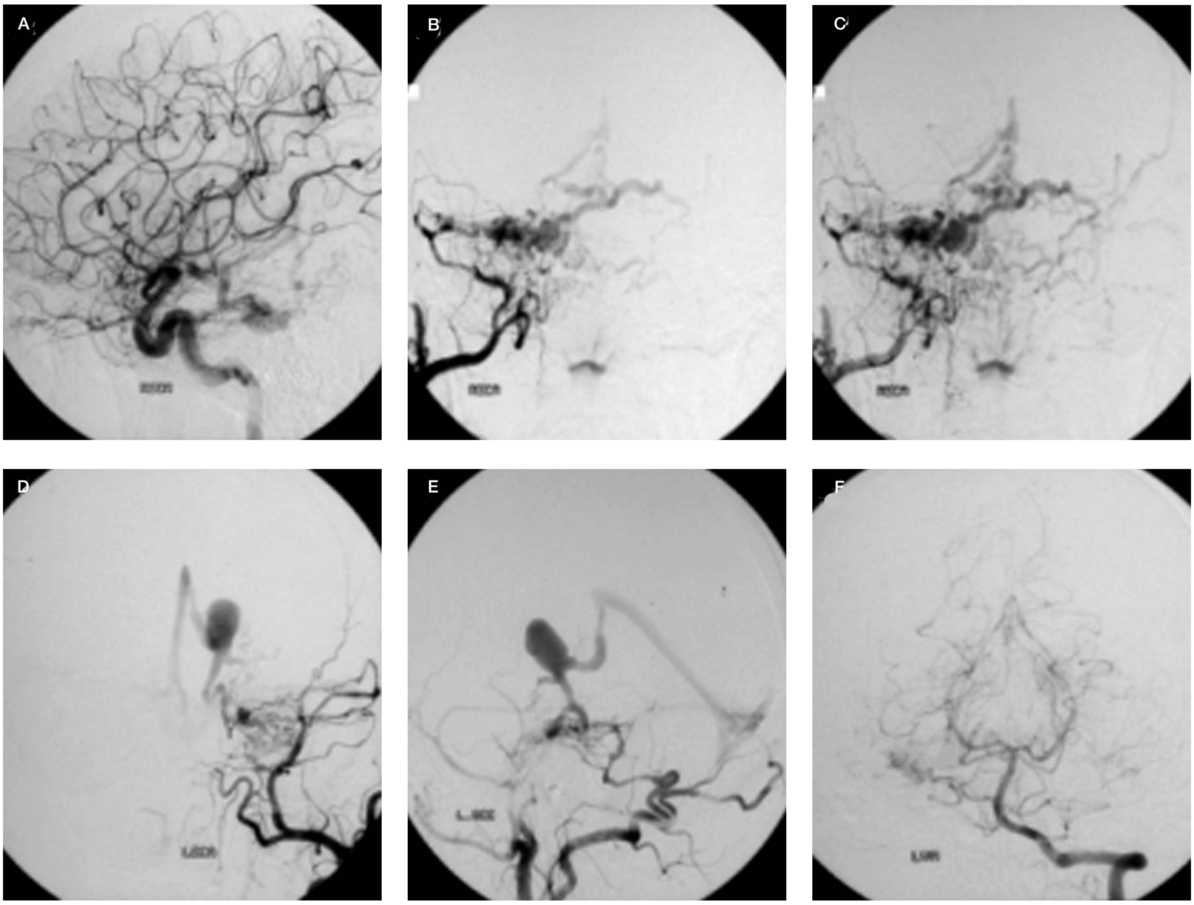

Figure 2 A) Right internal carotid angiogram (lateral view) demonstrates a dilated basal tentorial C5 branch (meningo-ophthalmic variant) supplies the dural arteriovenous malformation. B) The early phase of the right distal external carotid angiogram (AP view) shows a dural arteriovenous malformation of the petrosal apex fed by petrosal and petro-squamosal branches of middle meningeal artery. There is immediate filling of the dilated petrosal vein with a venous aneurysm, which drains to the anterior ponto-mesencephalic vein and basal vein of Rosenthal. C) The late phase of the right distal external carotid angiogram (AP view) shows dilated pial veins in the bilateral cerebellar and right cerebral hemispheres. D) Left distal external carotid angiogram (AP view) demonstrates a dural arteriovenous malformation of the petrosal apex fed by petrosal and petrosquamosal branches of the middle meningeal artery and drained directly to the perimesencephalic vein, supratentorially, eventually to the vein of Galen and to the straight sinus. A dilated venous aneurysm is noted at the junction of the lateral mesencephalic and posterior mesencephalic veins. E) Left occipital arteriogram (lateral view) shows a left petrosal dural arteriovenous malformation supplied by the mastoid branches of the occipital artery. F) Left vertebral angiogram (AP view) shows supply to the right side lesion by subarcuate artery from right anterior inferior cerebellar artery.

vertebral artery (subarcute artery from the anteroinferior cerebellar artery and artery of falx cerebelli).

The petrosal vein, the initial venous outflow, was connected to the anterior pontomesencephalic vein. It drained into the bilateral posterior mesencephalic veins. Extensive reflux to the cortical veins of the bilateral cerebellar hemispheres and to the right basal vein of Rosenthal was present, causing marked dilatation of the cortical veins. A smaller venous aneurysm was seen at the petrosal vein (figure 2).

Treatment and Follow-up

Since the venous aneurysm on the left side was an alarming size, it was decided to treat the left dural AVM first. Selective embolization of the middle meningeal artery supplies was done. The squamopetrosal branch was catheterized using a microcatheter (Prowler 10, Cordis, Miami, FL) obtaining flow arrest. It was embolized with 25% N-butyl cyanoacrylate (NBCA)-ethiodol mixture. The transmastoid branch of the occipital artery and the second pedicle of

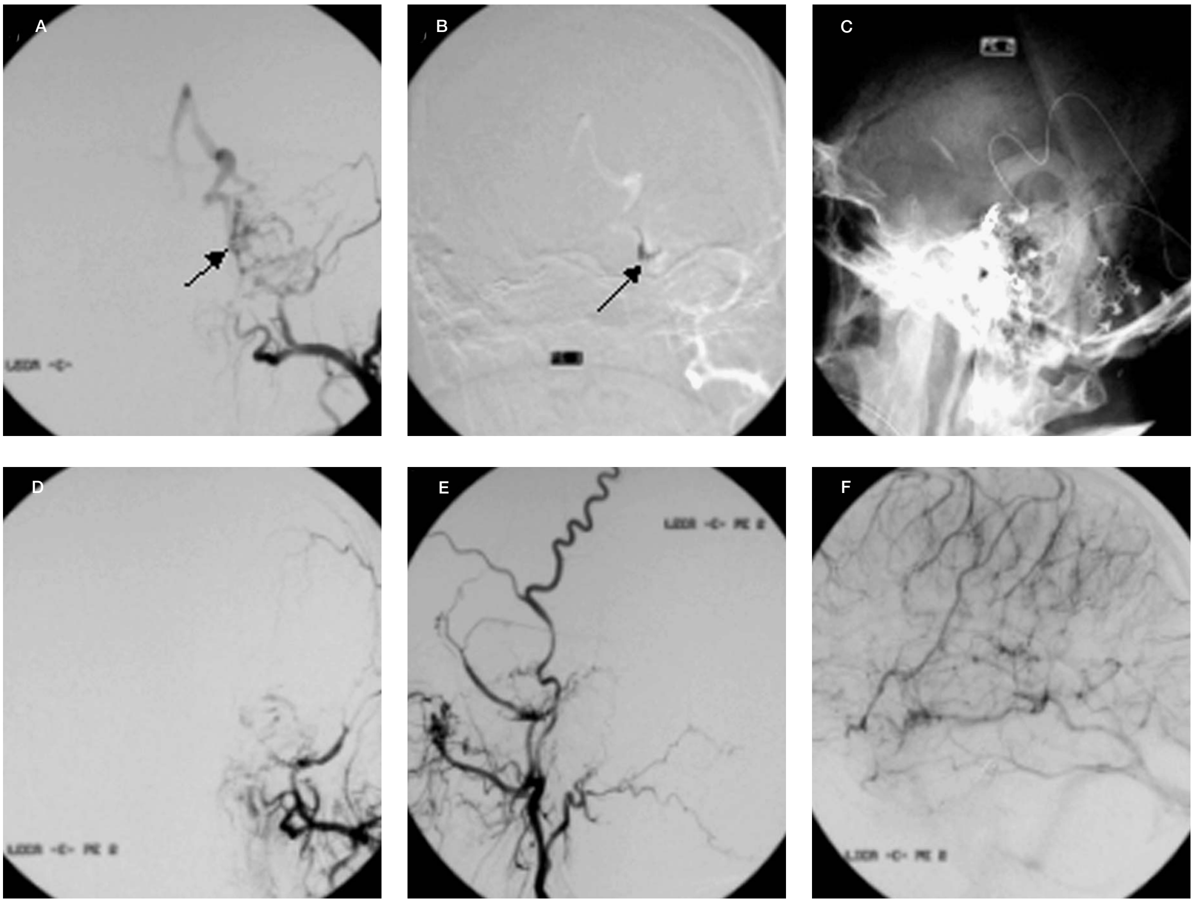

Figure 3 A) Left external carotid angiogram (AP view) before transvenous coil embolization shows recanalization of the previously embolized feeders from the middle meningeal artery. Note the site of AV fistula (arrow). B) Road map image shows coil loops cross the fistula (arrow). C) Plain film (lateral view) shows portion of a microcatheter via transjugular approach and coils prior to detachment. D,E) Post-embolization angiogram of the left external carotid trunk (AP and lateral view) shows complete occlusion of the dural AVM. Feeding pedicles decrease in caliber, and there is stagnation of the contrast. F) On the venous phase of the final angiogram of the left internal carotid artery, the basal vein of Rosenthal is now filling antegradely to the vein of Galen and straight sinus

the squamopetrosal branch were embolized with NBCA-ethiodol mixture similarly. It penetrated to the fistulas. The remaining feeders could not be catheterized to use liquid embolic material safely. These were closed with polyvinyl alcohol (PVA) particles, 250-500 microns in size.

The internal carotid artery supply was not attempted. The CT scan on post-embolization day two showed partial thrombosis of the left side venous aneurysm. Five days later, the right side lesion was embolized. The supplies from the hypoglossal, occipital and middle meningeal arteries were embolized with 20% NBCA-ethiodol mixture.

The patient underwent right retrosigmoid craniectomy for complete disconnection of residual shunts on the right side on the second post embolization day. Following surgery, initial neurological deficits were completely recovered, however, the patient developed permanent hearing loss on the right side. The cause was not clearly explained. It was decided to follow the patient instead of a second craniectomy to cure the left side dural AVM.

The patient complained of increasing intensity of tinnitus 17 months later. He was reassessed by selective angiogram, which demonstrated recanalization of the feeders embolized with PVA particles and increased size of the venous aneurysm. It was decided to attempt transvenous occlusion of the residual AVM. A

6 F guiding catheter (Envoy, Cordis, Miami, FL) was placed in the left jugular bulb and a microcatheter (Prowler 14, Cordis, Miami, FL) was advanced to the petrosal vein. The catheter tip could not be passed through the fistula to the arterial side. The initial attempt to close the draining vein with a 5 mm loop coil (Orbit coil; Trufill Detachable Coil System [DCS] Orbit™ coils, Cordis, Miami, FL) failed to secure the coil mesh in the initial portion of the draining vein. The catheter was repositioned again close to the fistula and a 6 mm diameter coil was advanced and two first loops crossed the fistula. Slowing down of flow was noticed immediately and complete occlusion of shunting was confirmed on the angiogram after completion of coil deployment.

The final angiogram of the left internal carotid artery showed complete occlusion of the all supplies. The basal vein of Rosenthal was now filling antegradely to the posterior mesencephalic vein, vein of Galen and straight sinus (figure 3). The patient did not have any new deficits and has been followed clinically. Pulsatile tinnitus has been completely eliminated.

Discussion

The etiology of dural AVMs of the tentorium is unclear. Most patients with tentorial dural AVMs are in the fifth and sixth decades of life, the superior petrosal sinuses are not visualized, and numerous tiny arteries supply the dural shunts. This clinical and anatomical configuration suggests that tentorial dural AVMs are acquired lesions like other dural AVMs [11]. There is no report of tentorial dural AVMs arising as a result of trauma or surgical procedure. Interestingly, tentorial dural AVMs occur more than twice as often in men than women; this male predominance is similar to the anterior cranial fossa dural AVMs.

Venous drainage is the determinant of clinical manifestation as documented by many publications [8,12]. Unlike dural AVMs of the transverse-sigmoid or cavernous sinuses that drain into venous sinuses directly, tentorial dural AVMs drain primarily into the pial veins. Dural AVMs that drain directly into a venous sinus without pial reflux have low rates of hemorrhage or neurological deficits. The thick sinus walls are resistant to rupture from elevated venous pressure and patients with these AVMs typically present with symptoms of bruit caused by increased flow through these arterialized sinuses [13,14]. In contrast, the dural AVMs that drain directly into pial veins are associated with extremely high rates of hemorrhage [2,3,15]. Hemorrhage occurs either from cortical veins or venous aneurysms. Venous outflow obstruction of the parent (draining) sinus contributes to the arterialization of thin-walled pial veins, which increases venous pressures and promotes the formation of venous aneurysms, thereby increasing the potential for hemorrhage [16]. Furthermore, patients may develop neurological deficits or seizures due to venous hypertension [8,17-19]. Some studies illustrated hemorrhage rates of 58 to 74% and aggressive neurological behavior of 79 to 92% in cases of tentorial dural AVMs [1-5,9-10].

In cases of tentorial dural AVMs, treatment should aim at complete cure to prevent hemorrhage or neurological deterioration.

Transarterial embolization alone is rarely successful because some feeding vessels are too small to undergo selective embolization with liquid embolic material or because there is a risk of infarction in cranial nerves such as the facial nerve and lower cranial nerves IX-XII. Even though decreasing arterial flow to the dural AVM reduces the effects of venous hypertension, the risk of hemorrhage remains unchanged if the dural AVM is not entirely obliterated [5,9]. Pre-operative embolization reduces the arterial supply and the occluded vessels provide a good intraoperative road map for understanding the surgical anatomy [9].

Transvenous embolization is useful for dural AVMs associated with the major dural sinus. However, a transvenous approach for deep-seated dural AVMs such as our case is difficult because the initial venous drainage flows entirely through pial veins. In selected cases, it is possible to treat tentorial dural AVMs with ectatic deep venous drainage via the transvenous route [6,20].

Percutaneous transvenous therapy of tentorial dural AVMs can be performed safely when a venous drainage pouch that is separate from veins draining normal brain can be identified and accessed with a microcatheter. As seen in our case, the draining veins of the petrosal ridge dural shunt are the petrosal vein, and confluent draining vein of the brain stem and cerebellum. These are long and tortuous routes through fragile pial veins to reach the site of fistulas, unlike cavernous or transverse-sigmoid dural AVMs. Therefore extremely careful manipulation and catheterization are needed to avoid possible rupture of a draining vein. Once the fistula is reached, detachable coils, either fiber or bare platinum, are effective to block the initial part of the draining vein. In our case, we were able to cross the fistula with a couple of loops of coil into the arterial side and the shunt was closed immediately.

Some authors do not think there is a role for stereotactic radiosurgery in the treatment of dural AVMs [21]. Radiosurgery of dural AVMs has been reported infrequently but with good results with complete occlusion in 44-87% of cases and minimal morbidity [22-29]. Radiosurgery can be well applied for tentorial dural AVMs because they are deep seated and usually have a multitude of tiny vessels feeding a small plexiform nidus. The combined use of stereotactic radiosurgery and transarterial embolization can enhance the effectiveness of this technique and reduce the risk of worsening symptoms during the follow-up period [9,24,27]. The goal of radiosurgery is to eliminate the remaining feeding arteries that are too small or cannot be embolized without a risk of infarction [9].

Even though endovascular procedures have become a first-line treatment for dural AVMs, surgery offers additional strategies [9]. Since venous outflow is usually a single vein, surgical excision of a network of fistulas is unnecessary and exposure of the venous drainage and surgical occlusion alone can achieve a cure [5,7,21,30-34]. Venous occlusion does not exacerbate venous hypertension unless thrombosis extends in the normal venous drainage [30]. Direct surgical obliteration was recommended in the following circumstances: if the ruptured aneurysm is not obliterated by means of embolization; if the feeding arteries are too small to embolize; if a dural AVM persists after stereotactically guided radiation therapy is administrated and the patient is symptomatic; or if there is significant risk of radiation injury to the surrounding structures [9,35].

After completion of the imaging studies, treatment plans for deep-seated dural AVMs begin with consideration of transarterial embolization of the feeding arteries from the external circulation. After transarterial embolization, the patient may be treated with stereotactically guided radiation therapy or direct surgical obliteration. Combining endovascular embolization with stereotactically guided radiation therapy or surgery in a staged manner has proven to be safe and effective for the treatment of deep-seated dural AVMs.

Conclusions

Petrosal tentorial dural AVMs are a unique subset of intracranial dural AVMs that are prone to hemorrhage due to retrograde pial venous drainage or associated venous aneurysms. They require definitive treatment because of their potential risk of bleeding and neurological deficits. These dural AVMs are difficult to obliterate by endovascular embolization alone because of the multiple tiny feeding arteries that are adjacent to vital normal vascular beds and difficulty in access of a transvenous route. Reducing arterial flow by transarterial embolization and obliterating the remaining dural malformation with radiosurgery or direct microsurgery should be the best treatment strategy. Transvenous coil embolization could be attempted in limited cases. We present the first case of bilateral petrosal ridge dural AVMs completely treated by a combination of endovascular and surgical treatment.

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